stasis is present may have as their cause gastro-intestinal stasis. Pyelograms should be made in cases of persistent pyelonephritis to demonstrate the absence or presence of ureteral stricture.

I wish to express my grateful appreciation of the courtesy of Dr. Arthur B. Cecil, who has so kindly made the material for this study possible.

PERSONAL OBSERVATIONS ON UNUSUAL FORMS OF ACUTE POLIOMYELITIC PARALYSIS.*

With Remarks on Clinically Related Types of Epidemic Encephalitis and Landry's Paralysis

By WALTER F. SCHALLER, M. D., San Francisco

Wickman [1] has classified the following clinical forms of poliomyelitis:

1. The spinal poliomyelitis form.
2. The form resembling Landry's paralysis.
3. The bulbar or pontine.
4. The encephalitic.
5. The ataxic.
6. The polyneuritic (resembling neuritis).
7. The meningitic.
8. The abortive.

Weisenburg [2] in a study of 717 cases in the 1916 Philadelphia epidemic modified this classification as follows:

1. The spinal form.
2. The form resembling Landry's paralysis.
3. The pontine bulbar:
   - (a) Bulbar.
   - (b) Pontine.
   - (c) Pontine bulbar.
   - (d) Pontine spinal.
   - (e) Bulbar spinal.
   - (f) Pontine bulbar spinal.
4. Encephalitic.
5. Cerebellar.
6. Meningitic.
7. Abortive.

It will be seen that Weisenburg's classification differs more particularly in the subdivisions of the bulbar types and the omission of the so-called polyneuritic type. In fact, this author is convinced that the pain and tenderness in the affected limbs, which is so marked and characteristic of a certain group, is not at all of neuritic origin. He found at no time pressure over the nerve trunks producing pain. In favor of the view of the meningeal origin of these sensory disturbances is the rapid subsidence of pain following lumbar puncture. In fact, from the standpoint of therapy lumbar puncture was considered indicated and was done in every patient on admission to the hospital and was repeated as occasion arose according to meningeal symptoms and pain.

In the spinal types Weisenburg makes the point that the progression of the paralysis in both upper and lower limbs is from the proximal portions to the distal portions of the limbs. He makes the statement that there is no record of a single instance in which the paralysis started in the parts below the knee and extended upward. In the recession of the paralysis the distal portions cleared up more rapidly than the proximal. A more rapid improvement in the upper extremities took place when contrasted with the lower extremities. A Landry type is spoken of, but is not considered to be as distinct as is commonly supposed.

Wickman classified Landry's paralysis as a form of poliomyelitis. From a study of two unpublished cases of Landry's paralysis—one in which a necropsy was done and the spinal cord and peripheral nerves studied—it is my personal opinion that it is very doubtful whether this is the case.

Charles K. Mills [3] in 1910, studying an outbreak of poliomyelitis in the Lehigh Valley and Philadelphia of that year, mentions cases reported in Norway and Sweden and in this country showing lesions practically identical with those in the cord, also in the cortex and in the basal ganglions. He speaks of the so-called cerebello rubro-spinal symptomatology—coarse tremor with some paresis and rigidity; the whole somewhat like paralysis agitans, or a mixture of this disease and multiple sclerosis. Although on the lookout for this form, Dr. Mills had evidently not met with an example of it.

This brings up the question of the possible identity of epidemic infantile paralysis and epidemic encephalitis, which has been suspected because of the similarity of the histo-pathological picture. The fact that this latter disease has always been associated in epidemic form with influenza; that it is unusual for it to produce lower motor neuron paralysis and atrophies comparable to poliomyelitis; that adults are predominantly affected; that the seasonal prevalence is different; and that no corresponding increase in the number of cases of infantile paralysis occurred when the encephalitis epidemic was at its height: all these would speak against this identity.

Strauss,[4] Loewe and Hirshfeld claim to have demonstrated a filterable virus in epidemic encephalitis, to have cultivated it and by it to have reproduced the disease in animals.

Although morphologically this organism resembles that described by Flexner and Noguchi in poliomyelitis, it acts differently in its infectiousness in different animals, namely, in monkeys and rabbits. Strauss told me personally this summer that he believes that the virus of influenza and epidemic encephalitis are the same.

Thalhimer [5] confirms the above authors on the specificity of this organism.

Riley [6] and others before him have discussed the spinal forms of epidemic encephalitis. Such a classification may lead to the impression that this clinical picture of encephalitis, and poliomyelitis are similar. This has not, however, been my experience. In a study of some twenty-nine personal cases of epidemic encephalitis gotten together over a year ago, and read before the annual meeting of the Pacific Railway Surgeons, no example of a frankly similar picture to acute poliomyelitic paralysis occurred. Riley describes an irritative type and a paralytic type, this latter often being an end result of the former, and included as a type largely on theoretic grounds.

* Read before the San Francisco County Medical Society Meeting of November 1, 1921.

The irritative type is evidenced either by myoclonic or fibrillary contractions.

In the last number of the Archives of Neurology and Psychiatry (November, 1921) Morris and Jacobson [7] have written an excellent article on acute ascending myelitis of the infectious type, with necropsy findings in two cases. The literature on this type is reviewed; they do not feel that they can conclude definitely that these cases are due to infection by the poliomeylitic virus, although it is equally difficult to deny it.

It may be said, therefore, that there is definite proof lacking in the identity of acute poliomyelitis, epidemic encephalitis, and Landry's paralysis, although it still may be possible that they are different clinical expressions of the same disease. I note that in "Oxford Medicine" Peabody accepts as cause of poliomyelitis the organism of Flexner and Noguchi.

When we are dealing with cases of acute poliomyelitis the clinical forms are so variable that sometimes we are in doubt about the correctness of the diagnosis, particularly at the onset of the disease. I have recently met with two examples, both in adults, in which the first probability which occurred to my mind on seeing these patients was a transverse myelitis.

CASE REPORTS

Case I. F. G. E., male, age 21—Private Record No. 1801—Pre-paralytic onset of three days, commencing with weakness of the legs gradually increasing, and difficulty in starting flow of urine. No pain. Examination revealed a total flaccid paralysis of left lower extremity, and also of right lower extremity excepting some motion at the right ankle. A distended bladder extending almost to the umbilicus necessitated catheterization. Questionable hypesthesia below middle of thighs in both extremities. No disturbance of deep sensibility and no pain on deep pressure of muscles. A history of a penile sore, in spite of negative serological reactions, made it appear probable that this was a case of transverse myelitis on a syphilitic and endarteritic basis. Time showed the error of this view, as the paralysis remained a flaccid one; there never occurred any pathologic pyramidal tract signs; and there were no lasting sensibility disturbances. The muscles eventually showed frank atrophies and reaction of degeneration with tendency to recovery of function especially in the muscles of the right lower extremity.

Case II. B. G., male, age 34—Seen in consultation at the Southern Pacific Hospital, San Francisco, October 18, 1921. Pre-paralytic onset as follows: Eleven days before the paralysis violent pains on both sides of chest, accompanied by slight chill and a temperature of 103° F., made probable a diagnosis of pleurisy or pneumonia. Two days later the temperature fell to normal and the pain disappeared. Three days later the patient returned to his occupation and felt well with the exception of some pain in his chest on deep inspiration. Three days after return to work a return of pain, but at this time across the small of back, radiating down the anterior part of both thighs to the patellae. Pain was lancinating and intermittent and continued for three days. On the third day about 11 o'clock in the morning the patient was unable to cross his legs in bed, and this weakness progressed so that by 7 o'clock that evening he had no use of the lower extremities except to move the toes. The arms were not involved. The examination in consultation revealed a flaccid paraplegia with no discernible involvement of sensation. There was some pain on deep pressure of the affected muscles, but more so by stretching the muscles either by passive flexion or extension, and particularly by the Kernig maneuver. There was some motion left in the toes of both feet but no motion at the hips, knees or ankles. In this case, as the foregoing, patient was obliged to be catheterized twice after the onset of paralysis. This case is of such recent occurrence that marked atrophies have not yet developed.

Another clinical type which has interested me very much is the predominant type associated with distressing and continued pain in the affected muscles. We have investigated these cases by lumbar puncture to determine whether the meningeal reaction ran pari pasu with the subjective sensory disturbances.

Case III. R. C., male, age 18.—Stanford Dispensary No. 94194—Pre-paralytic symptomatology of nervousness, irritability, sleeplessness and diplopia following overwork. The paralysis of the extremities began in the left arm, extended to the left lower extremity, then to the right arm, and then to the right lower extremity. Whereas, objective sensation was quite normal in the affected extremities, the patient noticed from the beginning marked sensitiveness of the muscles especially on pressure. The examination showed a flaccid quadriplegia with hypotonia, areflexia and atrophy much more marked in the proximal than the distal parts of the extremities, although the patient was paralyzed to the extent that he could not assist himself or move from the supine position in bed.

The following were the lumbar puncture findings in this case under the following dates:

	Pressure	Leucocytes	Globulins
April 22, 1921.....	250 mm.	18.1 per cmm.	increased
April 26, 1921.....	?	33.7 "	"
May 2, 1921.....	180 mm.	10.9 "	"
May 11, 1921.....	85 mm.	23.1 "	"
May 18, 1921.....	150 mm.	10.9 "	"
May 24, 1921.....	.....	18.6 "	"
June 2, 1921.....	145 mm.	28.1 "	"
June 9, 1921.....	.....	126.8 "	"
June 23, 1921.....	140 mm.	10.6 "	"
July 5, 1921.....	70 mm.	8.1 "	"

Following the puncture of May 24, June 1, and June 9, mercurialized autogenous serum was injected in order to stimulate the leptomeninges to greater effort to overcome the apparent subacute infection of the subarachnoid spaces. When the patient left the hospital on July the 12th the pain in the muscles although less was still present.

Case IV. A. N., female, age 11—Stanford Dispensary No. 97459—Entered hospital with complaint of paralysis of both lower extremities from the hips down. Duration about two weeks. Prodromal symptoms of "growing pains" in the legs, especially in the calves, and some headaches; increasing pain in the legs and in the lumbar region, with chilly sensations. Two days after this onset fell in attempting to walk and afterwards inability to use the lower extremities. Immediately prior to the paralysis, sensation of numbness and tingling in the feet. Examination: Both lower limbs held flexed on the abdomen, and legs flexed on the thighs, with some motion in the right toes. Exquisite tenderness of muscles on manipulation; areflexia, hypo-tonicity. Sensation was unaffected. The examination was made exceedingly difficult on account of the great muscle tenderness. Active motion was very difficult to determine on this account, although the flexion posture of the limbs suggested that more active motion was possible than could be elicited.

A lumbar puncture on this patient done two days after admission showed a considerable in-

crease in the globulins and a leucocyte count of 11.8. On account of the faulty position of the lower extremities in flexion and the possibility of contractures, the orthopedic surgeons were called in consultation. On September the 1st, or about three weeks after patient was admitted, a lumbar puncture was done under great difficulty, the fluid being contaminated by an admixture of blood. There were but 10.5 leucocytes per cu. m. m. with some increase in the globulin, which might have been accounted for by the normal blood content of white cells. This count was, however, not satisfactory. This patient left the hospital after the last puncture and has not since been heard from.

Case V. S. R., male, age 3½—Stanford Dispensary No. 98753—Pre-paralytic symptoms lasting three days, commencing with headache and a temperature of 102° F. The fever fell on the second day, but the child was languid and rather stuporous. The third day he was unable to stand because of paralysis of the right leg. This was on September the 20th. Examination showed flaccid paralysis of the right leg in extension, the leg being externally rotated and the foot everted. The tendon reflexes of this extremity were not elicited. No evident objective sensory disturbances. The left leg showed no paralysis. A very striking feature of this case was pain and tenderness on passive motion or pressure of the affected leg muscles, and also of the left leg muscles, but to a considerably less extent. The spinal fluid examinations were as follows:

	Leucocytes	Globulin
September 23, 1921.....	32	positive
September 27, 1921.....	11.2	"
October 12, 1921.....	48	"
October 29, 1921.....	3.1	negative

When last seen (October the 29th) the power of flexion and extension of the right thigh was possible, but no motion was present at the knee or in the toes. The tenderness was decreasing but still present.

It occurred to us to seek some references regarding the spinal fluid content following acute poliomyelitic paralysis. Whereas Draper gives no data concerning the fluid after the first few days, Ruhrah and Mayer state that "cells disappear rapidly so that after two weeks the count is either normal or nearly so."

This is probably the case in most instances, but in our series the persistence of the fluid reactions most probably indicated a meningeal origin of pain in the third and fifth cases, the former showing an increase in the cells for more than two months, and the third not clearing until one month. Unfortunately, the most typical case of pain, or the second case, we were not able to thoroughly study from the standpoint of meningeal reactions.

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(909 Hyde Street.)

STANFORD COLLOQUIA AT THE SAN FRANCISCO HOSPITAL

Surgical — (By Dr. Sterling Bunnell) — Our first patient is a middle-aged Mexican woman, with extensive tubercular enlargement of the cervical glands of all of the triangles in the left side of the neck. There is an especial enlarged one, probably with abscess, under the upper part of the sternocleidomastoid muscle. She noticed enlarged glands here for six years, though the greatest enlargement has taken place in the last month; this probably from abscess formation. Pathology of this degree is beyond cure by X-ray. I feel that X-ray and hygiene is the best treatment for beginning tubercular adenitis.

Surgery of this condition has gained ill repute because often merely the enlarged glands, and not the gland bearing tissue, has been removed from the neck. Considering this, it is not at all surprising that recurrent cervical enlargements are so frequent after operative removal. It is my conviction that the proper way to remove the glands and gland bearing tissue is en bloc, just as we do for carcinoma. It has been my experience that whenever the whole tubercular gland bearing mass has been removed in one piece, without tearing the tissue and spreading the germs about, that that patient has been cured, providing that the tonsils are removed and that there is no other tubercular focus. In such cases I have never seen local recurrence. A block dissection was then done and because of the magnitude of the operation it was followed by a transfusion of 900 cc. of blood. This reduced the pulse from 140 to 90.

The next patient is a woman forty years of age and very fat, who has had typical attacks of gall stones for six years. The present attack has lasted a week and was accompanied by a temperature of 101. About the lower part of the abdomen is a huge roll of fat, which suggests the necessity for a lipectomy, but peculiarly enough the layer of free peritoneal fat is as scanty as in a thin person. This, together with the finding of an infantile uterus, suggests hypopituritarism. The gallbladder is somewhat enlarged and congested, slightly thicker walled and more gray than normally and adherent to the neighboring viscera. The surface of the liver in the immediate vicinity shows a ribbing of scar tissue and along the cystic duct and common duct can be felt several enlarged glands. The pancreas is slightly harder than normal. Many stones can be felt in the gallbladder. In this case we have a full set of signs of cholecystitis. Quite often, however, we have a good history of cholecystitis and no gross signs in the gallbladder, except perhaps the enlargement of the telltale gland along the cystic duct. In such a case it is best to be guided by the history and remove the gallbladder. . . .

Our next case is that of an injury to the knee joint, caused by sitting upon it with the leg in the abducted position. There is local tenderness over the internal lateral ligament and pain is produced when the leg is abducted. There is also a greater degree of lateral motion in the injured leg compared with her normal leg. It